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EMOTIONAL PROCESSING IN FUNCTIONAL NEUROLOGICAL DISORDER: A REVIEW, BIOPSYCHOSOCIAL MODEL AND RESEARCH AGENDA

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ABSTRACT

Functional neurological disorder (FND) is a common and highly disabling disorder, but its aetiology remains enigmatic. Conceptually, there has been reduced emphasis on the role of psychosocial stressors in recent years, with a corresponding increase in neurobiological explanations. However, a wealth of evidence supports the role of psychosocial adversities (e.g., stressful life events, interpersonal difficulties) as important risk factors for FND. Therefore, there is a need to integrate psychosocial (environmental) and neurobiological factors (e.g., sensorimotor and cognitive functions) in contemporary models of FND. Altered emotional processing may represent a key link between psychosocial risk factors and core features of FND. Here, we summarise and critically appraise experimental studies of emotional processing in FND using behavioural, psychophysiological and/or neuroimaging measures in conjunction with affective processing tasks. We propose that enhanced preconscious (implicit) processing of emotionally-salient stimuli, associated with elevated limbic reactivity (e.g., amygdala) may contribute to the initiation of basic affective/defensive responses via hypothalamic and brainstem pathways (e.g., periaqueductal grey). In parallel, affect-related brain areas may simultaneously exert a disruptive influence on neurocircuits involved in voluntary motor control, awareness, and emotional regulation (e.g., sensorimotor, salience, central executive networks). Limbic-paralimbic disturbances in patients with FND may represent one of several neurobiological adaptations linked to early, severe and/or prolonged psychosocial adversity. This perspective integrates neurobiological and psychosocial factors in FND and proposes a research agenda, highlighting the need for replication of existing findings, multimodal sampling across emotional response domains and further examination of emotional influences on sensorimotor and cognitive functions in FND populations.

INTRODUCTION

Functional neurological (conversion) disorder (FND) is defined by the presence of neurological symptoms (e.g., sensorimotor, cognitive) that are not explained by identifiable neurological pathology.[1] FND can present similarly to almost any neurological illness (e.g., epilepsy, stroke, Parkinson's disease) and represents a notable proportion of neurology outpatient referrals.[2] FND often results in severe and/or chronic symptoms with considerable impact on patients' social/occupational functioning, in addition to significant healthcare and societal costs.[3] Nevertheless, there exists ongoing inconsistency in diagnostic classification and terminology for the disorder and its subtypes.[1, 4] Box 1 outlines the terminology used throughout this article.

Box 1. Terminology and abbreviations

FND = functional neurological disorder

FND-seiz = FND with seizures

FND-movt = FND with abnormal movements (e.g., tremor, gait, dystonia)

FND-par = FND with paralysis/paresis

Additional terms for FND in the literature reviewed: conversion disorder, dissociative (neurological) disorder, psychogenic (neurological disorder)

Additional terms for FND-seiz in the literature reviewed: non-epileptic attacks/seizures, dissociative seizures/convulsions, psychogenic non-epileptic seizures

Biopsychosocial frameworks acknowledge the variety of predisposing (e.g., psychosocial adversity, gender, physical illness, exposure to symptom/illness models), precipitating (e.g., physical injury, mental health symptoms, interpersonal conflict, other stressors), and perpetuating (e.g., avoidance, illness beliefs/expectations, social isolation) factors that can contribute to FND.[3, 5, 6] However, the exact mechanism(s) underlying FND symptoms are still not fully understood and there is no well-accepted explanatory model. Contemporary explanations have moved away from psychodynamic trauma-focused models and instead emphasise dysfunction of higher-order cognitive processes.[7-9] Nevertheless, the importance of psychosocial adversity in FND cannot be underestimated given that rates of early-life and proximal adverse events have been

repeatedly found to be higher in FND samples relative to controls.[10] Stressful life-events including abuse/neglect, ongoing relationship disturbances, occupational stress and caring responsibilities are commonly reported.[6, 11-13]

Patients with FND also frequently report difficulties in emotional functioning, including anxiety, depression, alexithymia and/or affect dysregulation.[3, 14, 15] Greater psychosocial adversity and/or affective dysfunction are associated with poorer quality of life, greater symptom severity, reduced resilience, elevated dissociation, and poorer prognosis in FND samples.[16-25] Psychological interventions (e.g., cognitive behavioural therapy) seeking to identify interactions between emotions, unhelpful thoughts/behaviours, psychosocial difficulties and FND symptoms show emerging efficacy in treating FND,[26] underscoring an important role for altered emotion emotional processing in this population.

There has been increasing research interest in emotional processing in FND in recent years, moving beyond self-report measures to controlled experimental task-based studies. Here, we critically review the experimental literature on emotional processing in FND to date, including only studies using behavioural, psychophysiological and / or functional neuroimaging measures. We then discuss the implications of these data for the pathophysiological basis of FND and propose an agenda for future research.

EMOTIONAL PROCESSING IN FND: A NARRATIVE REVIEW

We aimed to identify published experimental research studies on emotional processing in FND since 1965. We searched Embase, PsychInfo, and Medline/PubMed databases using the following terms: conversion disorder, functional neurological, hysteria/hysterical, psychogenic, non*epileptic seizures, dissociative seizures, combined with 'emotion*' and 'affect*'. Studies involving the presentation of emotionally-valenced stimuli (i.e., pleasant/appetitive or unpleasant/aversive) combined with the measurement of at least one response domain (i.e., subjective/behavioural, psychophysiological, neuroimaging) within laboratory settings were included. Studies using only self-report measures and/or qualitative techniques were excluded, as were case reports/series and conference proceedings. The

identified studies and key findings are summarised below and in Supplementary Table 1.

Behavioural & psychophysiological findings

Facial expression processing

Increased preconscious (implicit) attentional biases for angry and/or happy faces have been observed in people with FND-seiz.[27-29] The attentional bias for angry faces correlated positively with sexual abuse history and basal cortisol levels,[27, 28] whereas attentional bias for happy expressions was positively associated with seizure frequency in another study.[29] Emotional facial expressions also disproportionately disrupted ongoing cognitive processing in people with FND-seiz, including working memory[30] and task-switching performance.[31]

There is some evidence of altered explicit (conscious, intentional) facial emotion processing in people with FND. Reduced accuracy for recognising emotional facial expressions and reduced phasic skin conductance responses (SCRs) was observed in FND-seiz participants in one study.[15] Altered reaction times for explicit identification of sad and happy faces have also been reported in children and adolescents with FND, [32] alongside elevated heart rate (HR) and reduced heart rate variability (HRV) in the same sample.[33] Additionally, avoidant behavioural responses to angry faces have been observed in FND-seiz.[34] Three studies, however, did not report differences in accuracy for facial expression identification in people with FND, suggesting the need for further research.[32, 35, 36]

Theory of Mind / mentalising

A small number of studies indicated possible 'Theory of Mind (ToM)' or mentalising deficits in FND populations.[36, 37] Impaired 'affective ToM' was observed in a sample with mixed-symptom FND,[37] and an overall pattern of 'over-mentalising' has been reported in people with FND-seiz.[36] The latter tendency to 'over-mentalise' was positively associated with perceived stress in the previous month in that group.[36]

Affective picture paradigms

Altered subjective, psychophysiological, motor and/or somatosensory responses have been observed in FND samples during exposure to affective images from the International Affective Picture System [38] (IAPS).[39-43] Different samples or subgroups of individuals with FND displayed elevations in either subjective or somatic (autonomic, reflexive) responses to IAPS images, but not elevations in both together,[41-43] suggesting diminished integration of conscious and bodily emotional experiences.

Possible interactions between emotional and somatomotor responses during processing of IAPS stimuli have also been identified. Blakemore et al,[39] for example, found evidence for facilitated manual output force in people with FND-movt whilst viewing negative IAPS images. Fiess et al.[40] reported increased FND symptoms and reduced discomfort threshold after intentional emotion regulation during IAPS exposure. These findings indicate a possible amplifying effect of negative emotion and/or intentional emotion regulation on somatosensory processing.

Emotional learning and memory

There is preliminary evidence for deficits in instrumental learning in people with mixed FND symptoms, observed using a monetary reward/loss paradigm.[44] However, an earlier study found that prior associative conditioning of auditory stimuli with emotional faces had no effect on subsequent performance on a subjective agency task involving the same auditory stimuli.[45] Further research on associative and instrumental learning is necessary to expand on these initial findings.

There is as yet no evidence for declarative emotional memory impairment in FND samples. Brown et al.[46] tested memory for emotional and neutral words in people with FND-par, reporting an overall deficit on word recall that was not specific to the emotion condition. Furthermore, Aybek et al.[47] noted no between-group differences on behavioural responses for personalised life event reminders in people with FND-par.

Elevated affective (autonomic) arousal

Elevated autonomic arousal at baseline or during affective processing has been observed using varied psychophysiological measures.[15, 27, 33, 34, 41-43] Findings include elevated salivary cortisol,[34] HR,[33] skin conductance levels,[15] and SCRs,[41] and lower HRV,[27, 33, 42]. In addition, potentiated startle responses have been observed in patients with FND-movt.[43]

Summary - behavioural and psychophysiological findings

FND is associated with altered social-emotional cognition, including an implicit attentional bias towards facial emotion and disrupted behavioural and autonomic responses. Baseline hyperarousal, altered autonomic responses, and discordance between subjective and objective responses during affective processing have been demonstrated. Preliminary evidence suggests possible instrumental learning deficits and abnormal sensory-motor responses during affective processing tasks.

Task-based neuroimaging studies

Figure 1 summarises the neural regions in which alterations have been most consistently observed during affective processing in people with FND, along with possible functional connectivity differences.

[INSERT FIGURE 1]

Facial expression processing

Altered activation has been frequently observed in affect-related regions during facial expression processing tasks. Three studies reported increased amygdalar activation for one or more emotionally-valenced facial expressions.[48-50] Interestingly, in one study, elevated amygdala activation was seen only during passive movement of the affected limb in patients with FND-par.[49] Increased activity has also been reported in several other limbic-paralimbic regions across different FND samples, including the periaqueductal grey (PAG), dorsolateral prefrontal cortex (dlPFC), parahippocampal gyrus and cingulate cortex / paracingulate gyrus.[35, 50, 51] However, decreased orbitofrontal cortex (OFC), insular and parahippocampal gyrus engagement was noted in patients with FND-seiz in some contrasts.[35]

Another pattern observed commonly during facial emotion processing is altered activity in motor regions. For example, increased activity has been reported in the supplementary motor area (SMA) / premotor cortex,[50] precentral gyrus,[35] and cerebellum.[52] Conversely, reduced activations in the precentral gyrus, putamen and cerebellum have also been noted in other contrasts.[35, 51] A particularly important finding is greater functional connectivity between the amygdala and motor circuits during facial emotion processing tasks.[48, 49]

Affective picture paradigms

Alterations in limbic/paralimbic and motor circuit activity (i.e., PFC, posterior cingulate, insula, amygdala, hippocampus, cerebellum, putamen) have also been reported in several studies during exposure to IAPS stimuli.[39, 40, 52, 53] Furthermore, increased amygdala - middle frontal gyrus functional connectivity has also been demonstrated.[51]

Emotional learning and memory

Amygdalar hyperactivation has been reported during monetary losses in people with FND, alongside a trend towards diminished dlPFC engagement.[44] Patients with FND-par also exhibited increased motor, temporal and dlPFC activity during exposure to illness-relevant life event reminders in the study by Aybek et al.[47] This same sample also displayed increased connectivity between amygdala and motor areas (SMA) during the experiment.

Summary – neuroimaging findings

The most consistent findings are increased amygdala reactivity, heightened motor circuit activation, altered prefrontal engagement, and enhanced motor-limbic circuit functional connectivity during affective processing tasks across several FND subgroups.

DISCUSSION

Altered social-emotional cognition

There is considerable evidence for impaired social-emotional cognition in individuals with FND. The best supported behavioural finding is an implicit/preconscious attentional bias towards emotional facial expressions in people with FND-seiz,[27-29] which is linked to adverse life events,[27] hypothalamic-pituitary-adrenal (HPA-axis) dysfunction,[28] and symptom severity.[29] These behavioural data link directly to elevated amygdalar activity observed during implicit facial expression processing,[48-50] which together suggest enhanced affective salience of these stimuli.

Increased recruitment of a subcortical ‘unconscious’ processing stream (e.g., thalamo-amygdala pathway,[54] along with initiation of basic affective responses via the hypothalamus and PAG, could arguably lead to this automatic and rapid attentional allocation to such stimuli. Reduced thalamic volumes[55, 56] and elevated PAG activation[50] reported in FND samples provide additional support for this suggestion. Furthermore, Perez et al.[57] noted that individual differences in amygdala and PAG volumes correlated positively with mental health symptoms, trait anxiety, and role limitations due to affective disturbances in a mixed FND cohort. Together, these findings support an overlap between structural and functional alterations in the pathophysiology of FND.

There is some evidence for explicit facial expression processing differences in both children and adults with FND, linked to prior psychosocial stressors and/or maladaptive attachment styles.[15, 32] These behavioural findings are supported by emotion processing alterations in regions such as the amygdala, OFC and insula.[35, 48-50] Differences in facial expression processing have also been noted in other somatic symptom disorders.[58, 59] The studies identifying impaired mentalising in FND samples[36, 37] further point towards difficulties in accurately inferring others’ mental states and emotional experiences.

These alterations in social-emotional cognition, if replicated, may be important features in future theoretical perspectives and psychological interventions for FND.

Impaired interoceptive awareness

A discrepancy between subjective (cognitive) responses and psychophysiological measures during emotional processing was observed by several investigators,[41-43] suggesting reduced integration between conscious emotional experience and somatic responses. Visceromotor responses to emotional stimuli may not be accurately perceived or interpreted by people with FND, representing a failure of 'interoception'. Another study, which examined neuroendocrine responses to social stress, also indicated discordance between subjective and neuroendocrine stress responses in patients with FND-par.[60] There is also preliminary experimental evidence for interoceptive deficits on the classic heartbeat detection task in patients with FND.[61]

A potential neurobiological basis for interoceptive deficits in FND may involve the insula. In the studies reviewed here, the insula was less engaged during incidental processing of neutral facial expressions in people with FND-seiz[35] and during unpleasant/neutral IAPS images in patients with FND-movt.[52] In addition, the insula has been implicated in the broader structural and functional neuroimaging literature on FND.[62-65] Furthermore, anterior insular volumes correlated negatively with symptom severity in patients with mixed FND.[21, 57]

Poorer performance on measures of interoceptive accuracy has also been described in individuals with other somatoform disorders[59, 66] and training in interoceptive accuracy can lead to reduced somatic symptoms in these patients.[67] Together, these findings highlight the potential for transdiagnostic interoceptive deficits across FND and somatic symptom disorders.

Hyperarousal and autonomic hyper-reactivity

Several studies identified elevated baseline autonomic arousal and HPA-axis activation in people with FND, and others noted similar elevations during experimental emotional processing tasks or in phasic responses to affective stimuli.[15, 27, 33, 34, 41-43]

These findings relate to early studies in people with FND, demonstrating more spontaneous electrodermal fluctuations and reduced habituation of SCRs to acoustic probes.[68, 69] Furthermore, cardiac-related autonomic measures show that sympathetic activity is elevated at baseline or pre-ictally in people with FND-seiz, but that it reduces during and/or after seizures,[70, 71] revealing a possible function of the seizures as a means of reducing heightened physiological / affective arousal, of which some patients are subjectively aware.[13, 72] Elevated HR and reduced vagal tone have also been observed in FND-movt.[73] Furthermore, Bakvis et al.'s[34, 74] findings concur with other reports of altered HPA-axis functioning in people with FND,[60, 75] with two of the studies demonstrating positive associations between HPA-axis markers and adverse life events.[60, 74]

The observed hyperarousal and increased autonomic reactivity in FND is likely to be related to hyperactivity and diminished habituation of the amygdala observed during affective processing tasks.[48-50] This profile resembles findings in several other neuropsychiatric disorders and may represent a 'limbic scar' resulting from early or chronic psychosocial stress.[76, 77]

Similar increased baseline or task-based autonomic/HPA activation have also been observed in other somatic symptom disorders.[59, 78] There is also evidence of reduced pituitary volumes in both individuals with FND and health anxiety.[79, 80] Collectively, these studies draw attention to elevated arousal and autonomic reactivity as an important pathophysiological feature that may be associated with altered social cognition, psychosocial adversity, and vulnerability to other somatic symptoms.

Increased motor circuit activity and limbic-motor system connectivity

Increased activation in motor regions during affective processing has been commonly observed across affective tasks in this review.[35, 39, 40, 47, 50, 52, 53] These findings overlap with previous reports of a range of structural alterations in motor neurocircuits in FND subgroups. Cortical atrophy, for example, has been reported in premotor and motor areas in people with FND-seiz,[81] the former associated with depressive symptoms in that group. In contrast, Aybek et al.[82] reported cortical thickening of the premotor area in FND-par, and differences in coping through accepting responsibility (i.e., an adaptive response) has been associated with

individual differences in ventral premotor cortical thickness in mixed FND patients.[83] SMA volumetric increases were noted in young people with FND, with SMA volumes negatively correlating with reaction times for facial emotion identification.[84] The SMA is thought to be involved in the selection of action sequences in response to internal cues, whereas the premotor area is involved in action selection in response to external cues.[85] The cerebellum, which has also been reported to demonstrate group-level volumetric differences in patients with mixed FND[57] and FND-seiz,[81] has well-documented roles in coordinating motor-cognitive-affective processing.[86]

Increased limbic-motor circuit connectivity has also been observed during affective processing tasks[47-49, 51] and at rest[35] in FND samples. It is possible that enhanced limbic-motor coupling might mediate the influence of emotion on voluntary motor control in FND, possibly by contributing to automatic activation or inhibition of motor sequences.[87] Routes by which this influence might occur potentially involve the insula, ACC, or ventral striatum. Additionally, evidence exists for direct projections from the amygdala to voluntary motor cortex in humans and animals[88] and whilst these pathways are generally thought to be less dense than other fronto-limbic pathways,[89] it is possible that they could be structurally or functionally stronger in people with FND.

Altered emotion regulation / prefrontal activity

Differences in several prefrontal regions during affective processing tasks have been identified,[39, 44, 47, 50, 51] including the dlPFC and cingulate cortex / paracingulate gyrus.

The dlPFC is implicated in the cognitive regulation of emotional states,[90, 91] and the dorsal anterior cingulate cortex (ACC) is known to be important for allocation of cognitive control and regulation of sensorimotor/autonomic affective reactions.[92] Altered prefrontal engagement during these tasks, therefore, might represent disturbances in the effortful allocation of resources directed towards emotion regulation.

Prefrontal cortex (PFC) regions are closely connected with the amygdala and PAG, so elevated limbic activation could potentially disrupt PFC functioning through

reciprocal interactions. Additionally, neuroendocrine stress responses (i.e., glucocorticoid and pro-inflammatory release), mediated by the amygdala and hypothalamus might also adversely impact on the functioning of PFC emotion regulation systems. Nonetheless, more research is needed to understand the specificity of PFC activation profiles in the pathophysiology of FND.

Summary: the possible role of emotional processing in generating and perpetuating FND symptoms

Figure 2 presents an overview of emotional processing alterations in FND and how they may contribute to the generation and perpetuation of FND symptoms. In this model, we propose that a range of vulnerabilities might predispose towards altered emotional processing in people with FND, and these factors are likely to interact and vary between individuals.

[INSERT FIGURE 2]

Emotional processing alterations in FND include:

- Limbic (amygdalar) hyperactivation, excessive affective (autonomic) arousal and threat-related hypervigilance.
- Impaired interoception of visceromotor emotional responses, possibly promoting reduced emotional awareness and insufficient integration of affective, cognitive, and viscerosomatic experiences.
- Suboptimal emotional regulation, possibly including under- and over-regulation at distinct instances in the same individual.
- Disturbances in explicit (subjective) interpretations of affective stimuli (e.g., emotion recognition and mentalising).

Altered emotional processing in patients with FND could disrupt awareness and/or higher-order control of a range of other cognitive, behavioural and somatic processes, thereby contributing directly to FND symptom generation, as follows:

- Limbic hyperactivation and autonomic hyperarousal facilitates activation of learned action sequences (i.e., FND-movt; FND-seiz) and/or innate affective

responses (e.g., autonomic arousal, tonic immobility, psychomotor agitation; FND-seiz; FND-par), via limbic afferents to voluntary motor regions and PAG areas respectively.

- Diminished subjective awareness of one or more lower-level processes (e.g., sensation, behaviour, motor responses) is mediated by disturbed connectivity between limbic regions and those involved with awareness of self, body and behaviour (e.g., cingulo-insular, temporo-parietal regions).
- Altered ('top-down') control of sensorimotor and affective responses, possibly mediated by altered PFC-amygdala interactions, results in the perceived lack of agency/voluntary control that patients with FND experience.

Methodological considerations

There are several methodological concerns when interpreting the literature including:

- Sampling
 - Most studies included HC comparison groups only, limiting the extent to which the findings can be viewed as specific to FND.
 - FND samples often included comorbid psychiatric disorders, which are common in FND, but few studies adjusted for mood and/or anxiety potentially obscuring result specificity.
 - Small sample sizes ($n < 20$) were common, particularly in neuroimaging studies, limiting statistical power and confidence in the results reported.
 - Many studies focused on only one subgroup of FND patients, which does not fully encompass the range of sensory-motor symptoms common in many with FND.
 - No studies to date have directly compared FND-subtypes using emotion processing paradigms.
- Psychotropic medications are rarely accounted for, yet such medications (e.g., antidepressants, anxiolytics, antiepileptic drugs) likely influence mood, cognition and emotional responsivity.
- Neuropsychological measures of cognitive abilities relevant to task demands have not been routinely utilized.

- ‘Reverse inference’[93] is often used in interpretation of neuroimaging findings as evidence for emotional processing disturbances, often in the absence of subjective, behavioural or psychophysiological alterations.

Future directions

This review points towards several important avenues for future research, as follows:

1. Replications of existing findings are needed across a broader range of FND symptom types, with additional experimental paradigms and larger samples.
2. Inclusion of both clinical and non-clinical control groups with similar risk factors to FND, such as controls who have experienced trauma and/or mild-moderate anxiety and depression.
3. There is a need for multimodal sampling of emotional processing responses, rather than relying on only behavioural, psychophysiological, neuroendocrine or neuroimaging measures.
4. Relationships between relevant comorbid symptoms (e.g., post-traumatic stress disorder, depression, dissociation), psychosocial risk factors (i.e., trauma, life events) and emotional processing alterations in FND should be clarified.
5. Future studies should further examine interoceptive awareness in FND samples.
6. Emotional processing should be studied beyond the laboratory setting, to enhance the ecological validity of findings.
7. Additional work is needed to explore the developmental trajectories of emotional processing alterations and associated neurobiological processes in paediatric FND samples.
8. The intersection of emotion processing, motor control, agency perception, and somatic sensations should be explored in more detail.

CONCLUSIONS

The literature to date indicates heightened preconscious (‘bottom-up’) processing of emotionally significant stimuli, increased affective arousal, disrupted

'top-down' regulation, and altered interoception of bodily emotional responses in people with FND. Furthermore, there is evidence of limbic and motor system hyperactivation, and enhanced interaction of these neurocircuits, during emotional processing in FND samples. We propose that these alterations in emotional processing could contribute directly to the generation of FND symptoms, through enhanced limbic influence on a range of neural circuits involved in awareness and control of multiple lower-level processes, including sensory, motor, and behavioural functions. These emotional processing differences might arise from a variety of biological and psychosocial risk factors, including but not limited to aberrant neuroplasticity of cortico-limbic circuits associated with early or prolonged psychosocial adversity. This perspective integrates neurobiological and psychosocial processes in a unified model of FND and indicates clear directions for future research.

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Competing interests:

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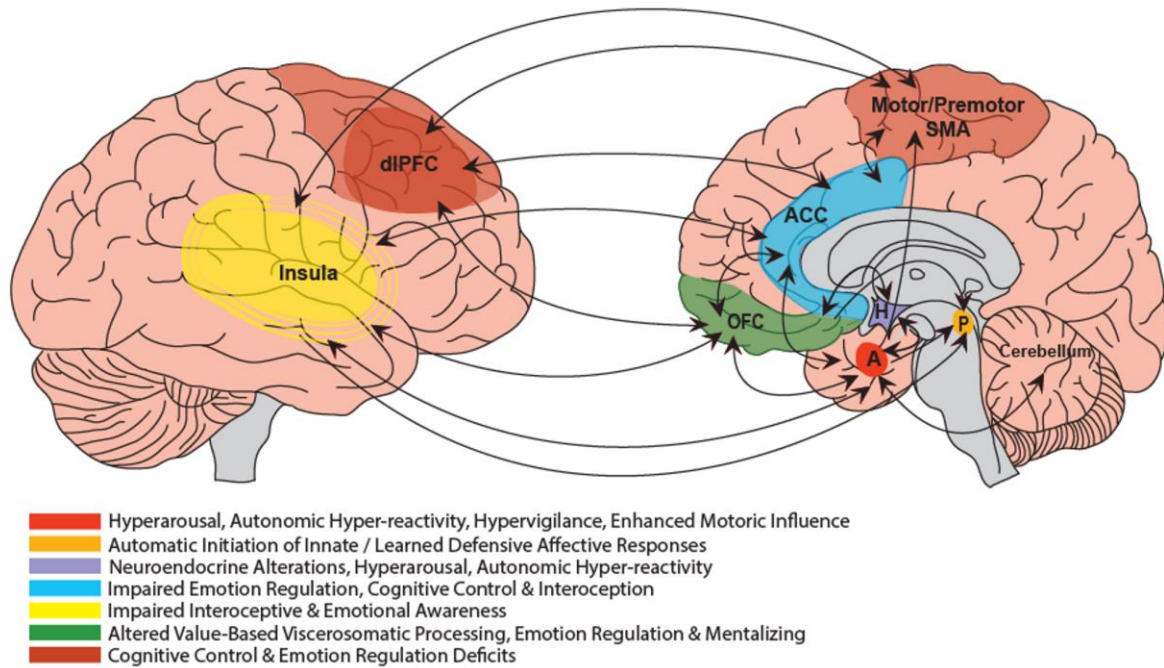


Figure 1. Schematic representation of key regional abnormalities and emerging functional connectivity alterations in FND during emotional processing. Main findings from the literature include heightened limbic (amygdalar) and motor (SMA/PMA/M1, cerebellum) activations; enhanced limbic-motor network functional connectivity; altered prefrontal and paralimbic (ACC, dlPFC, OFC, insula) engagement and elevated functional connectivity with the motor system. **Key:** A = amygdala; ACC = anterior cingulate gyrus*; dlPFC = dorsolateral prefrontal cortex; H = hypothalamus; OFC = orbitofrontal cortex*; P = periaqueductal grey; SMA = supplementary motor area; *both ACC and OFC project to periaqueductal grey and hypothalamus.

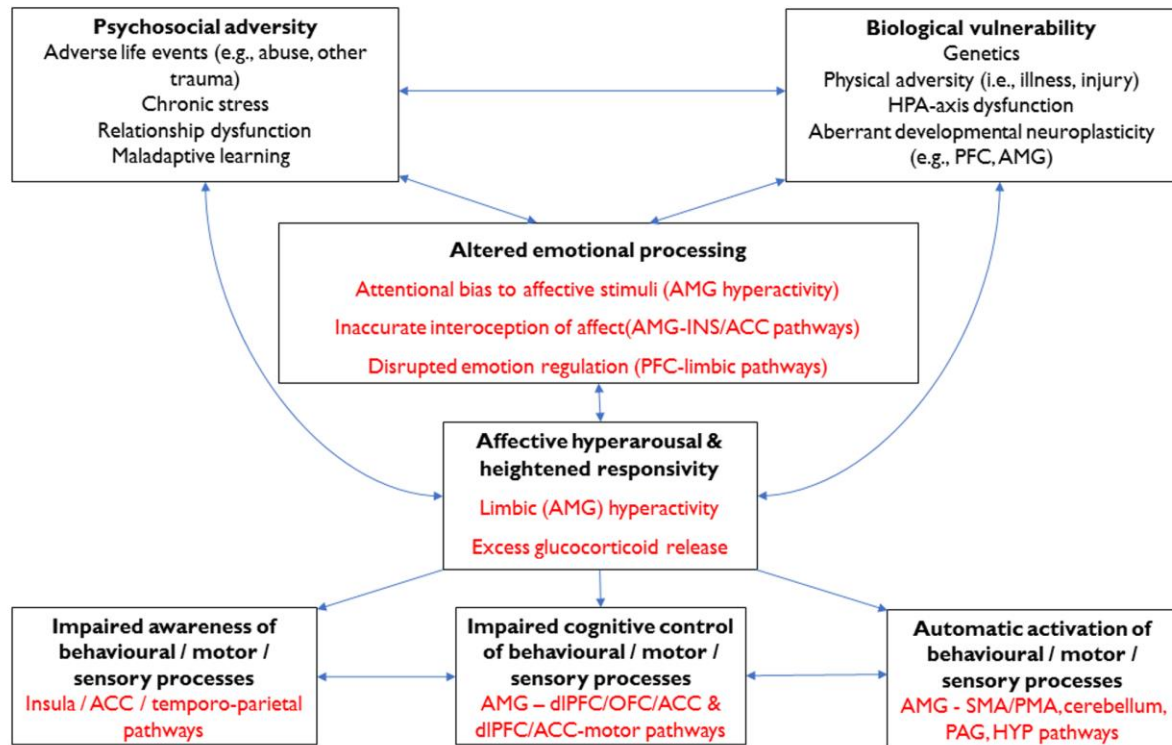


Figure 2. A summary of the possible role of emotional processing in generating (and perpetuating) functional neurological symptoms. Key emotional processing differences include an attentional bias to affective stimuli, inaccurate interoception, and suboptimal emotional regulation. Affective hyperarousal and hyper-reactivity are linked to elevated limbic (amygdalar, HPA-axis, PAG) activity which exerts a disruptive influence on neurocircuits crucial for cognitive control, initiation of behavioural/motor responses, and awareness. **Key:** ACC = anterior cingulate gyrus; AMG = amygdala; dIPFC = dorsolateral prefrontal cortex; HPA = hypothalamic-pituitary-adrenal; HYP = hypothalamus; INS = insula; OFC = orbitofrontal cortex; PAG = periaqueductal grey; PFC = prefrontal cortex; PMA = premotor area; SMA = supplementary motor area.

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Supplementary Table 1. Experimental studies of emotional processing in FND grouped by task type

FACIAL EMOTION PROCESSING / SOCIAL COGNITION TASKS				
Authors	Participants	Methods	Key findings	Limitations
Aybek et al. [1]	FND-par (n = 12), HCs (n = 14)	Implicit facial expression processing (sad, fearful, neutral): manual gender discrimination, 2s exposure	No group differences on behavioural measures (i.e., gender discrimination)	No report of medication use or discussion of possible medication effects
	Diagnosis / recruitment: neurologist & neuropsychiatrist, neurology / neuropsychiatry outpatients Inclusion criteria: DSM-IV FND criteria, chronicity < 2 years, preceding stressor (during 12 months before illness onset) Exclusion criteria: psychosis, neurological disorder, non-fluency in English, unsatisfactory task performance Groups matched for: age, gender, estimated IQ, HADS scores, history of abuse	Event-related fMRI Primary dependent measures: BOLD responses: block (Fear-Neutral, Sad-Neutral), ROI (bilateral amygdala), whole brain analysis, RTs and accuracy for gender discrimination Additional measures: HADS; semi-structured clinical interview – elicited sexual abuse history Cognitive tests: NART	ROI: <ul style="list-style-type: none"> increased activity in left amygdala in FND-par group for sad and fearful faces lack of habituation in left amygdala for fearful faces (sensitisation) Whole brain: <ul style="list-style-type: none"> FND-par group increased activity for emotion (vs neutral) in PAG and frontal clusters (left dlPFC, bilateral SMA/premotor/left superior and medial frontal gyrus/left cingulate cortex) Effects remained after controlling for HADS Depression and/or Anxiety scores	Lack of clinical comparison group
Bakvis et al. [2, 3]	FND-seiz (n = 19), HCs (n = 20), ECs (n = 17)	Emotional Stroop paradigm – backwardly masked facial expressions (anger, happiness, neutral, 14ms exposure)	Increased AB scores for angry faces at baseline in FND-seiz group (HCs and ECs showed negative AB scores for angry faces); remained significant when controlling for age	Several Ps had mood/anxiety disorders but no data provided on severity of anxiety/depression
	Diagnosis/recruitment: neurologist, v-EEG recording of typical seizure, specialist epilepsy centre Inclusion criteria: v-EEG diagnosis of FND-seiz, seizures involving partial/complete LoC, ≥ 2 seizures in previous 12 months Exclusion criteria: antidepressants, corticosteroids, lithium, beta-	Task completed before / after stress induction (Trier Social Stress test) Primary dependent measures: attentional bias (AB) scores (RTs for colour-naming of expressions minus those for neutral faces), colour-naming errors, heart rate,	Positive correlations of AB scores for angry faces (baseline) with sexual abuse (TEC) scores [27] and basal cortisol levels [28] in FND-seiz group FND-seiz group performed worse (more errors) at baseline than during stress	Unclear whether AED use was excluded from FND sample Findings may not generalise to FND-

	blockers, cimetidine, ketoconazole, neurological disorder, endocrine disorder, history of epileptic seizures	HRV, blood pressure, salivary cortisol	induction (controls performed comparably across conditions)	seiz patients who do not experience LoC
	Groups matched for: education, gender, contraceptive use, menstrual cycle, but FND-seiz group older and reported more interpersonal trauma than HCs	Additional measures: TEC, subjective anxiety (Likert scale), MINI Cognitive tests: Stroop interference	No group effects on Stroop scores FND-seiz group - lower HRV at baseline/recovery	
Bakvis et al. [4]	FND-seiz (n = 19), HCs (n = 20) Diagnosis/recruitment: as described in above section [2,3] Inclusion criteria: as above [2,3] Exclusion criteria: neurological disorder, endocrine disorder, history of epileptic seizures, unsatisfactory task performance, failure on effort test, insufficient saliva samples Groups matched for: age, gender, education, smoking, contraceptive use, menstrual cycle, but FND-seiz group had higher SCL-90-R Anxiety and Depression scores and more likely to be taking psychotropic medication (relative to HCs)	Emotional N-Back test: working memory task requiring participants to manually respond to a target letter in a sequence, either 0, 2 or 3 letters back (varying WM load), simultaneous presentation of emotional faces (neutral, happy, angry) or no distractor Test completed before and after stress induction (Cold Pressor test, hand immersed in ice cold water) Primary dependent measures: errors (omission / commission), RTs, salivary cortisol, blood pressure, subjective pain and anxiety (visual analogue scale, 0-100) Additional measures: SCL-90-R (Anxiety, Depression scales), MINI, Amsterdam Short Term Memory test (measuring effort)	Trend (p = .091) towards higher cortisol in FND-seiz group throughout experiment No group differences for blood pressure, subjective anxiety/pain, performance on the Cold Pressor test (duration hand immersed), RTs on N-back test FND-seiz group made more errors on the N-back test than HCs overall At baseline, FND-seiz group made more errors than HCs for the facial distractor conditions but not for no distractor condition After stress induction, the FND-seiz impairment on the N-back test was significant across all conditions Group effects on the N-back test remained significant when controlling for elevated SCL-90 Anxiety/Depression scores	Significantly more FND-seiz taking psychotropic medication, but not analysed / insufficient detail on type of medications
Bakvis et al. [5]	FND-seiz (n = 12), HCs (n = 20)	Approach-avoidance task: facial expressions presented (anger, happiness, 100ms exposure), Ps	FND-seiz group had higher cortisol levels throughout experiment	Several Ps had mood/anxiety disorder (although

	<p>Diagnosis/recruitment: as described in above section [2,3]</p> <p>Inclusion criteria: as described in above section [2,3]</p> <p>Exclusion criteria: as described in above section [2,3]</p> <p>Groups matched for: as described in above section [4]</p>	<p>asked to evaluate expression (forced choice) requiring flexion (avoidance) or extension (approach) of the arm</p> <p>Before and after stress induction (Cold Pressor test)</p> <p>Primary dependent measures: RTs (correct responses), errors, salivary cortisol</p> <p>Additional measures: SCL-90-R, MINI</p>	<p>FND-seiz group slower for anger-approach behaviour than for anger-avoidant behaviour (baseline)</p> <p>FND-seiz group slower for anger-approach than HCs (baseline)</p> <p>Effects remained after controlling for medication, anxiety/depression scores</p> <p>No group effects for happy faces</p>	<p>statistically controlled for SCL-90 Anxiety and Depression scores)</p> <p>Small sample size</p> <p>Tertiary recruitment setting</p>
Espay et al. [6]	<p>FND-movt (tremor) (n = 27), essential tremor (ET, n = 16), HCs (n = 25)</p> <p>Diagnosis/recruitment: clinical criteria, setting unspecified</p> <p>Inclusion criteria: published clinical criteria, tremor absent/minimal at rest</p> <p>Exclusion criteria: neurological disorder, severe anxiety/depression (HAM-A / HAM-D scores), using benzodiazepines, unsatisfactory task performance, unsuitable for scanning (e.g., obesity, prior neurosurgery), non-acceptance of diagnosis, malingering</p> <p>FND-movt and HCs matched for: age, gender, handedness, but FND-movt group shorter disease duration and higher HAM-A and –</p>	<p>fMRI</p> <p>Finger-tapping motor task: paced finger tapping with right or left hand</p> <p>Basic-emotion task: implicit facial expression processing (happiness, sadness, fear, neutral, 2s exposure), manual gender discrimination</p> <p>Primary dependent measures: BOLD responses, volumetric and connectivity analyses</p> <p>Additional measures: MINI, HAM-A, HAM-D</p>	<p>Finger tapping task: reduced cerebellar activity (VI) in ET compared to FND-movt group, during right finger tapping (controlling for HAM-D)</p> <p>Basic-emotion task (sadness vs neutral contrast, controlling for HAM-D):</p> <ul style="list-style-type: none"> FND-movt group showed increased activity in paracingulate gyrus and Heschel's gyrus compared to HCs FND-movt group showed reduced activation in right precentral gyrus relative to ET group <p>Volumetric analysis: FND-movt group showed smaller caudate volume and reduced grey matter in right postcentral gyrus, compared to HCs</p>	<p>Recruitment setting and diagnostician not clearly specified</p> <p>Psychiatric comorbidities present in sample (e.g., PTSD, panic disorder, social phobia, alcohol dependence)</p> <p>Possible medication effects (e.g., AEDs, antidepressants) not considered</p>

	D scores than ET group (and HCs for HAM scores)			
Espay et al. [7]	<p>FND-movt (dystonia) (n = 12), organic dystonia (OD) (n = 12), HCs (n = 25)</p> <p>Diagnosis/recruitment: neurologist, specialist movement disorder service</p> <p>Inclusion criteria: functional unilateral or asymmetric limb dystonia, clinical criteria / clinician consensus</p> <p>Exclusion criteria: no psychiatric or physical illness (HCs)</p> <p>FND-movt group younger, included more females, and higher HAM-D and HAM-A scores than OD group, FND-movt group shorter disease duration than OD group</p>	<p>fMRI</p> <p>Finger-tapping motor task: paced finger tapping with right or left hand</p> <p>Basic-emotion task: implicit facial expression processing (happiness, sadness, fear, neutral, 2s exposure), manual gender discrimination</p> <p>Primary dependent measures: BOLD responses, volumetric analyses</p> <p>Additional measures: MINI, HAM-A, HAM-D</p>	<p>Finger-tapping: no differences observed in BOLD response</p> <p>Basic-emotion task:</p> <ul style="list-style-type: none"> faces vs fixation contrast - the FND-movt group showed decreased activation in right middle temporal gyrus, bilateral precuneus; increased activation in right inferior frontal gyrus, bilateral occipital cortex, bilateral cerebellum, bilateral fusiform gyrus relative to HCs (pattern reported as similar in FND-movt vs OD analyses) emotional vs neutral faces contrast - differences between FND-movt and HCs observed for lateral ventricular white matter (right) and right fusiform cortex 	<p>Mood and anxiety disorder present in several FND-movt patients (although statistically controlled for HAM-A and HAM-D scores)</p> <p>Sample size small</p> <p>No behavioural test of emotional processing</p> <p>No detail on medication use</p> <p>FND-movt and OD groups not matched for age, gender or disorder duration</p>
Gul & Ahmad [8]	<p>FND-seiz (n = 72), HCs (n = 72)</p> <p>Diagnosis/recruitment: university hospital</p> <p>Inclusion criteria: DSM-IV criteria (clinical observation/history), seizure frequency ≥ 2</p> <p>Exclusion criteria: epilepsy (FND-seiz), psychiatric and neurological disorder, medication (HCs)</p>	<p>Task-switching experiment: Ps required to switch from age- to emotion-discrimination for happy and angry expressions</p> <p>Primary dependent measures: RTs, errors</p> <p>Additional measures: DASS, ERQ</p>	<p>Switch cost was higher for the age task than for the emotion task in the FND-s group, but not in the HCs</p> <p>FND-seiz group:</p> <ul style="list-style-type: none"> positive correlation between emotion suppression scores (ERQ) and switch costs negative correlation between cognitive reappraisal and switch costs 	<p>Lack of detail on diagnostic procedures and recruitment setting</p> <p>DASS scores not specified/controlled for</p> <p>Unclear whether other neurological illness or major psychiatric disorders were</p>

	Groups matched for: age, gender, education, SES (although statistical values unspecified), but more psychiatric diagnoses in the FND-seiz group			<p>excluded from FND-seiz group</p> <p>Ps aged 18-35 years only – unrepresentative</p> <p>No detail or exploration of medication effects</p>
Hassa et al. [9]	<p>FND-par (n = 13); HCs (n = 19)</p> <p>Diagnosis/recruitment: single neurologist, inpatient rehabilitation centre, diagnostic testing (i.e., MRI, motor evoked potentials, electromyography)</p> <p>Inclusion criteria: ICD-10 criteria for FND motor symptoms (hemilateral paresis), aged 20 years or more</p> <p>Exclusion: neurological disorder, PTSD, panic disorder, major affective disorder, psychosis, MRI contraindication</p>	<p>fMRI</p> <p>Emotional faces (sad, neutral) presented (1s duration) simultaneously with sensorimotor stimulation (passive movement of right/left hand or none), red dots superimposed on faces</p> <p>Primary dependent measures: BOLD response (ROI, amygdala, vmPFC, motor system), connectivity analyses (psychophysiological interactions), dot counting</p>	<p>No group differences on dot counting accuracy</p> <p>Left amygdala hyperactivation in FND-par group during sad faces/affected hand movement (passive)</p> <p>Increased functional connectivity of amygdala and pre-SMA, and amygdala-subthalamic nucleus in FND-par group vs HCs (sad>calm faces)</p> <p>No altered activity in limbic areas during emotionally neutral passive movement</p>	<p>Unclear whether groups matched for possible confounds (e.g., medication, psychopathology, gender, age)</p> <p>Lack of subjective and cognitive measures</p> <p>Dot-counting task showed a ceiling effect (100% accuracy in both groups)</p> <p>Inclusion of only one negatively valenced face condition (sadness)</p>
Kozłowska et al. [10]	<p>FND (mixed symptoms), paediatric (n = 57), HCs (n = 57)</p> <p>Diagnosis/recruitment: tertiary care, paediatrician, psychiatrist, psychologist</p>	<p>Facial expression recognition (explicit): expressions (anger, sadness, disgust, fear, happiness, neutral), 2s exposure, labelling task (selection of label from six options)</p>	<p>FND group had faster RTs for sadness and slower RTs for happiness, compared to HCs</p> <p>No group effects on accuracy</p> <p>DASS scores did not correlate with RTs or accuracy scores</p>	<p>Comorbidities common in sample (anxiety, depression, pain, behavioural disorder)</p>

	Inclusion criteria: DSM-IV-TR criteria	Primary dependent measures: recognition accuracy, RTs		No detail on medication and its possible influence on results
	Exclusion criteria: none specified	Additional measures: DASS		
	Groups matched for: age, gender, but DASS Anxiety, Stress and Depression scores elevated in FND sample			
Kozłowska et al. [11]	FND (mixed symptoms), paediatric (n = 57), HCs (n = 57)	Facial expression recognition (explicit): as described in above section [10]	FND group displayed: <ul style="list-style-type: none"> Intact behavioural performance on the auditory oddball task Impaired performance on the Go-NoGo task Elevated HR and reduced HRV at baseline and across tasks, including the emotional faces task (vs HCs) Lack of HR elevation during the faces task (vs HCs) Many findings driven by differences between adolescent FND Ps and controls 	Comorbid neurological/psychiatric diagnoses in a number of Ps, along with psychotropic medications (e.g., AEDs, antidepressants)
	Diagnosis/recruitment: as described in above section [10]	Auditory oddball task: discrimination of auditory target from auditory distractors based on pitch; measures selective attention		Observed effects not specific to emotional processing task
	Exclusion criteria: as described in above section [10]	Go-NoGo task: Ps required to inhibit a primed response to an infrequent 'NoGo' stimulus presented amongst more frequent 'Go' stimuli, measures behavioural inhibition		
	Groups matched for: as described in above section [10]	ECG	Autonomic measures did not correlate with DASS scores	
		Primary dependent measures: HR, HRV, respiratory rate		
		Additional measures: DASS		
Pick et al. [12]	FND-seiz (n = 40), HCs (n = 43)	Explicit facial expression recognition – labelling task (sadness, fear, anger, disgust, happiness, neutral, 6s exposure)	Significant group effect for recognition accuracy (FND-seiz scored lower), controlling for covariates (HADS, YoE) and medication status	FND-seiz patients without comorbid anxiety / mood disorder may be unrepresentative
	Diagnosis/recruitment: neurologist/neuropsychiatrist, outpatient neuropsychiatry services	Primary dependent measures: recognition accuracy, emotional		

	<p>Inclusion criteria: FND-seiz diagnosis based on video-EEG or clinical consensus (≥ 2 clinicians)</p> <p>Exclusion criteria: MDD, anxiety disorder, psychosis, neurological disorder, substance dependence</p> <p>Groups matched for: age, gender, handedness, ethnicity, SES, IQ and WMS-III Faces 1 scores, but FND-seiz group had fewer years of education and higher HADS (Anxiety and Depression) and IASC scores, compared to HCs</p>	<p>intensity ratings, SCLs (baseline, task), SCRs (amplitude, frequency)</p> <p>Additional measures: HADS, TEC, IASC</p> <p>Cognitive tests: WASI, BFRT, WMS-III Faces 1</p>	<p>Correlations: recognition of neutral faces with total trauma scores (TEC) and abandonment concerns (IASC)</p> <p>Elevated SCLs at baseline in FND-seiz group (controlling for HADS Depression and medication)</p> <p>Reduced SCR amplitude seen in FND-seiz autonomic responders ($n = 16$) vs control autonomic responders ($n = 16$), controlling for medication, anxiety, depression, education</p>	<p>Some FND-seiz patients not diagnosed with v-EEG</p> <p>Lack of clinical comparison group</p> <p>Tertiary recruitment setting</p>
Pick et al. [13]	<p>FND-seiz ($n = 38$), HCs ($n = 43$)</p> <p>Diagnosis/recruitment: as described in above section [12]</p> <p>Inclusion criteria: as described in above section [12]</p> <p>Exclusion criteria: as described in above section [12]</p> <p>Groups matched for: age, gender, handedness, ethnicity, highest qualifications, IQ, Stroop interference and BFRT scores</p> <p>FND-seiz group had higher scores on HADS (Anxiety, Depression), relative to HCs</p>	<p>Emotional Stroop: masked facial expressions (happy, angry, neutral), 17ms exposure, Ps required to colour name masking stimuli</p> <p>Primary dependent measures: attentional bias (AB) scores (RTs for emotion-neutral conditions), colour-naming errors</p> <p>Additional measures: HADS</p> <p>Cognitive tests: BFRT, WASI, Faces I (WMS-III), Stroop test</p>	<p>Increased AB scores in FND-seiz group (across happy and angry conditions), controlling for HADS and education</p> <p>Positive correlation between AB for happy faces and seizure frequency in FND-seiz group</p>	<p>Medication (AEDs) possibly impacted on findings</p> <p>Other limitations as described in above section [12]</p>
Schönenberg et al. [14]	<p>FND-seiz ($n = 15$), HCs ($n = 15$)</p> <p>Diagnosis/recruitment: neurologist, specialist epilepsy centre</p>	<p>Morphed facial emotion detection task (Ps indicate when they perceive emotion as expression morphs from neutral to emotional)</p>	<p>No group effect on facial emotion detection task</p> <p>More MASC errors in FND-seiz group (vs HCs)</p>	<p>7 Ps had a psychiatric diagnosis other than FND-seiz</p>

	<p>Inclusion criteria: video-EEG evidence of FND-seiz, ≥ 2 seizures in preceding year</p> <p>Exclusion: history of epilepsy, neurological disorder (all Ps), psychopathology (HCs)</p> <p>Groups matched for: sex, age, education level, PSS scores, but FND-seiz group had higher scores on TAS-20</p>	<p>Expressions included: anger, happiness, fear, sadness, surprise, disgust</p> <p>Theory of mind task: Movie for the Assessment of Social Cognition (MASC), mentalising about characters in a series of vignettes (dinner part setting)</p> <p>Primary dependent measures: intensity at which facial emotion detected, errors in facial emotion identification</p> <p>MASC - errors (dichotomous classification – under-mentalising, over-mentalising)</p> <p>Additional measures: MINI, TAS-20, PSS</p>	<p>FND-seiz group had an ‘over-mentalising’ pattern of errors overall</p> <p>Under-mentalising positively associated with TAS-20 scores in FND-seiz group</p> <p>Over-mentalising positively associated with PSS scores in FND-seiz group</p>	<p>Self-reported depression and anxiety not measured or controlled for</p> <p>Multiple correlations not corrected for familywise error (alpha of $p < .05$ used)</p> <p>No detail on medication and its possible influence on results</p>
Stonnington et al. [15]	<p>FND (mixed symptoms) (n = 29), Functional Somatic Symptoms (FSS, n = 30), Medical Controls (MCs, n = 30)</p> <p>Diagnosis/recruitment: various outpatient settings (primary, tertiary)</p> <p>Inclusion criteria: DSM-IV criteria, FSS (chronic fatigue, fibromyalgia, irritable bowel, etc), MCs (physical symptoms due to medical conditions), 18-60 years old</p> <p>Exclusion criteria: suicidality, substance abuse, cognitive</p>	<p>Frith-Happé Animations Task (AT): Ps describe what happened in a series of animations (random movement, goal-directed, ToM)</p> <p>Primary dependent measures: AT-L: emotional states attributed to shapes, LEAS scores</p> <p>Additional measures: AT-I (intentions attributed to shapes), Reading the Mind in the Eyes (RME) test (Ps infer affective states on the basis of</p>	<p>FND and FSS groups had lower scores on AT-L compared to MCs; this remained significant after controlling for PANAS Negative Affect and HAM-A Anxiety scores, but only the group effect for random movement was significant when controlling for MADR Depression scores</p> <p>No significant group differences on AT-I, LEAS, RME or MSS</p>	<p>No detail on medication use and its possible influence on results</p> <p>Diagnosis often based on clinical judgement</p>

	<p>impairment, psychosis, non-fluency in English</p> <p>Groups matched for: age, education SES, estimated IQ, current MDD, PTSD, trauma history, SCL-90 Somatic Symptoms scores, but significantly fewer males in FSS group</p> <p>Anxiety (HAM-A) and depression (MADRS) higher in FND and FSS groups relative to MCs</p> <p>Higher TAS-20 (FND vs MCs) and lower PANAS Positive Affect scores in FND and FSS groups vs MCs</p>	<p>eyes only), MSS (mental and physical state attributions), MINI, TAS-20, PANAS, MADR, HAM-A, SCL-90 Somatic Symptom scale, SF-36</p> <p>Cognitive tests: Wechsler Test of Adult Reading (estimated IQ)</p>		
Szaflarski et al. [16]	<p>FND-seiz (n = 12), ECs (n = 12), HCs (n = 24)</p> <p>Diagnosis: specialist epilepsy centre</p> <p>Inclusion criteria: video-EEG evidence of FND-seiz or temporal lobe epilepsy</p> <p>Exclusion: degenerative/metabolic disorders, history of TBI, pregnancy, recent suicidal behaviour, non-fluency in English, contraindication to MRI, abnormal MRI (medial temporal sclerosis not excluded)</p> <p>Groups (FND-seiz, ECs) matched for: gender, age, education, age at illness onset, seizure frequency</p>	<p>Implicit facial emotion processing task (happy, fearful, sad, neutral), manual gender discrimination, 2s exposure</p> <p>Event-related and resting-state fMRI</p> <p>Post-scan explicit facial expression recognition (labelling) test</p> <p>Primary dependent measures: behavioural (RTs, accuracy), BOLD (ROIs, connectivity, whole brain analyses)</p> <p>Additional measures: BDI-II, POMS</p>	<p>Accuracy did not differ between groups, but RTs longer for ECs compared to HCs and FND-seiz</p> <p>BOLD: widespread increased activation for emotional expressions in FND-seiz group, including:</p> <ul style="list-style-type: none"> • precentral gyrus (neutral, vs HCs and ECs) • superior/middle frontal gyri (happiness and neutral, vs HCs) • postcentral gyrus (happiness, vs HCs and ECs; neutral, vs HCs) • superior/middle temporal gyri (happiness, vs HCs; fearful, vs ECs) • parahippocampal gyrus (fearful, vs ECs) <p>FND-seiz group showed decreased activation in regions including:</p>	<p>Only patients with generalised motor FND-seiz included – potentially unrepresentative</p> <p>Possible medial temporal lobe sclerosis and AED use in the EC group - potential confounds</p> <p>Different acquisition protocols within same study</p> <p>Behavioural tests possibly underpowered due to sample size (n =</p>

	Illness duration longer in ECs, FND-seiz group scored higher on BDI-II than HCs and ECs, and higher on POMS total compared to HCs		<ul style="list-style-type: none"> • putamen (sadness, vs ECs and HCs) • cerebellum (happiness, vs HCs) • OFC (happiness, vs HCs) • insula (neutral, vs HCs) • parahippocampal gyrus (sadness, vs HCs) • cingulate gyrus (sadness, vs HCs) <p>FND-seiz group showed increased resting state functional connectivity of left/right amygdala with motor (thalamus, lentiform nucleus, cerebellum) and emotion processing (insula) circuits (vs ECs)</p> <p>Findings controlled for POMS scores and seizure frequency</p>	<p>12 in clinical groups)</p> <p>Behavioural test of facial expression recognition – possible ceiling effect due to pre-exposure of stimuli during scanning</p>
Voon et al. [17]	<p>FND-movt (n = 16), HCs (n = 16)</p> <p>Diagnosis/recruitment: ≥ 2 neurologists plus a psychiatrist, specialist movement disorder service</p> <p>Inclusion criteria: clinically definite FND-movt, no symptoms at rest, 19 years or more, diagnostic testing yielding negative results (i.e., MRI, nerve conduction)</p> <p>Exclusion criteria: TBI, antidepressants, neurological disorder, MDD, panic disorder, PTSD, substance abuse, other affective disorder or psychosis, MRI contraindication</p>	<p>Implicit facial expression processing (fear, happiness, neutral), 1s exposure, blocked fMRI design, manual gender discrimination</p> <p>Primary dependent measures: behavioural (RTs, accuracy), BOLD (ROI amygdala; functional connectivity)</p> <p>Additional measures: BDI, BAI, SCID-DSM-IV</p>	<p>No group effects on behavioural measures (RTs, gender discrimination accuracy)</p> <p>BOLD: Elevated right amygdala activation in FND-movt group (vs HCs) for happy-neutral contrast (and trend for fear-neutral), controlling for BAI/BDI</p> <p>Greater connectivity of right amygdala and right SMA in FND-movt group for happy-neutral and fear-neutral (vs HCs)</p>	<p>3 FND-movt Ps on nocturnal benzodiazepines and 1 on AEDs (although withheld the night before testing)</p> <p>Non-clinical control group only</p>

Groups matched for: age, gender, but BDI and BAI scores elevated in FND-movt group

AFFECTIVE PICTURE VIEWING TASKS

Authors	Participants	Methods	Key findings	Limitations
Blakemore et al. [18]	<p>FND-movt (n = 10), HCs (n = 10)</p> <p>Diagnosis/recruitment: neurologist, neurology outpatients</p> <p>Inclusion criteria: DSM-5 criteria for FND motor symptoms</p> <p>Exclusion criteria: neurological disorder, speech/hearing/visual impairments, MRI contraindications, recent psychiatric disorder (HCs)</p> <p>Groups matched for: handedness, maximum manual force, HADS scores</p>	<p>Emotional-force control task: precision grip contraction aimed at 10% of max force, combined with visual force output feedback or IAPS images (pleasant/unpleasant), 6s exposure, fMRI</p> <p>IAPS images presented again post-scanning</p> <p>Primary dependent measures: BOLD response, behavioural (mean force, coefficient of variation), post-scan subjective ratings of valence / arousal</p> <p>Additional measures: HADS</p>	<p>No group differences in post-scan subjective responses to stimuli (valence/arousal)</p> <p>Force output was higher in FND-movt group relative to HCs in the unpleasant condition; remained significant after controlling for age, baseline force and subjective reactions</p> <p>During unpleasant pictures (unpleasant > pleasant), FND-movt group showed less activation in PFC/IFG/insula and more activation in hippocampus and cerebellum, relative to HCs</p> <p>For regions correlated with force output (unpleasant > pleasant contrast), FND-movt group showed increased activation in hippocampus, amygdala, visual cortex, putamen, cerebellum (greater activation was observed in IFG and cerebellum in HCs)</p> <p>Distinctive emotional effects by group (unpleasant > pleasant contrast): FND-movt – cerebellum, left hippocampus, left posterior cingulate cortex, bilateral occipital gyrus (HCs – bilateral IFG, pre-SMA)</p>	<p>No explicit exclusion of comorbid psychiatric disorder or cognitive impairment in the FND-movt sample</p> <p>Medication effects not excluded – 6/10 Ps in the FND-movt group on medication (AEDs, antidepressants, anxiolytics or ≥ 1 of these)</p> <p>Post-scan subjective responses possibly influenced by within-scan exposure and/or small sample size</p>

Espay et al. [6]	<p>FND-movt (tremor) (n = 27), essential tremor (ET, n = 16), HCs (n = 25)</p> <p>Diagnosis/recruitment: as described in above section [6]</p> <p>Inclusion/exclusion criteria: as described in above section [6]</p> <p>Groups matched for: as described in above section [6]</p>	<p>fMRI</p> <p>Intense-emotion task: visual oddball paradigm with shapes (squares/circles), unpleasant pictures, and neutral pictures, Ps discriminate circles from all other cues</p> <p>Primary dependent measures: as described in above section [6]</p> <p>Additional measures: as described in above section [6]</p>	<p>No significant group differences in BOLD responses during intense-emotion task</p> <p>Functional connectivity: Increased connectivity between left amygdala and left middle frontal gyrus in FND-movt compared to HCs (controlling for HAM-D)</p>	<p>Pleasant images not included</p> <p>Additional limitations as described in above section [6]</p>
Espay et al. [7]	<p>FND-movt (dystonia) (n = 12), organic dystonia (OD) (n = 12), HCs (n = 25)</p> <p>Diagnosis/recruitment: as described in above section [7]</p> <p>Inclusion/exclusion criteria: as described in above section [7]</p> <p>Groups matched for: as described in above section [7]</p>	<p>fMRI</p> <p>Intense-emotion task: visual oddball paradigm with shapes (squares/circles), unpleasant pictures, and neutral pictures, Ps discriminate circles from all other cues</p> <p>Primary dependent measures: as described in above section [7]</p> <p>Additional measures: as described in above section [7]</p>	<p>Intense-emotion task - FND-movt group showed:</p> <ul style="list-style-type: none"> • decreased activation of left insula and left motor cortex (vs HCs and OD) • increased activation of left fusiform gyrus (vs HCs) • decreased activation of right opercular cortex and right motor cortex (vs OD) 	<p>Limitations as described in above section [7]</p>
Fiess et al. [19]	<p>FND (mixed negative sensory/motor symptoms) (n = 20), HCs (n = 20)</p> <p>Diagnosis/recruitment: neuropsychiatrist/neurologist (≥ 2), specialist rehabilitation setting</p> <p>Inclusion criteria: ICD-10 criteria, presence of ≥ 1 negative FND</p>	<p>Emotion regulation task: IAPS images (unpleasant, neutral), 2s exposure, Ps cued to passively observe (P) or regulate (R) reactions</p> <p>MEG</p> <p>Primary dependent measures: subjective symptom intensity change score, somatic sensation</p>	<p>MEG: HCs showed decrease in power when cued to regulate (vs when cued to watch passively), FND group did not show this preparation effect</p> <p>FND group showed greater overall emotion effect (negative-neutral) but no regulation effect (regulate-passive); HCs showed less pronounced emotion effect and significant regulation effect</p>	<p>SCL-90-R scores for HCs and group comparisons for the scale not presented</p> <p>No detail on medication and its possible influence on results</p>

	<p>symptom, normal or corrected vision</p> <p>Exclusion criteria: any history of CNS lesions/disorders</p> <p>Groups matched for: age, gender, years of education, but FND group had higher scores for SDQ-20, and TAS-26</p>	<p>(transcutaneous electrical stimulation, change in perception/discomfort threshold), stimulus-evoked power changes, subjective symptom intensity (Likert scale - 0-10)</p> <p>Additional measures: MINI (HCs), SDQ-20, SCL-90-R, TAS-26</p>	<p>HCs showed greater power decrease in bilateral frontal cortex during regulation (vs FND), FND group showed greater regulation effect in left central (sensorimotor) cortex (vs HCs) (controlling for presence of anxiety/depression)</p> <p>Correlation of bilateral frontal power (regulate) with FND symptom ratings (trend for sensorimotor regions)</p> <p>TENS discomfort threshold was lowered in FND group (vs HCs) after the emotion regulation task</p> <p>FND symptoms subjectively rated as more intense after the emotion regulation task (vs before)</p>	
Fiess et al. [20]	<p>FND (mixed negative symptoms, as above) (n = 21), HCs (n = 21)</p> <p>Diagnosis/recruitment: as described above [19]</p> <p>Inclusion criteria: as described above [19]</p> <p>Exclusion criteria: as described above [19]</p> <p>Groups matched for: age, gender, years of education, and handedness, but FND group had higher scores for SDQ-20, SCL-90-R Depression, PSSI and ETI</p>	<p>Passive viewing of IAPS pictures (pleasant, unpleasant, neutral), 333ms exposure</p> <p>Rapid serial visual presentation design</p> <p>MEG</p> <p>Primary dependent measures: global field power, ROI (posterior and central cortical areas)</p> <p>Self-report measures: SDQ-20, SCL-90-R, PSSI (PTSD symptoms), ETI (childhood trauma)</p>	<p>Reduced GFP, posterior and central power observed in the FND group 110-150ms post stimulus onset</p> <p>Left-hemisphere central modulation by stimulus category seen in the FND group but not HCs</p> <p>Effects not accounted for by SCL-90-R Depression, PSSI and ETI scores</p>	<p>No subjective/behavioural data reported</p> <p>Exclusion of seizures or patients with positive FND symptoms</p> <p>FND group undergoing intensive rehabilitation – sample possibly biased towards severity / chronicity</p> <p>No detail on medication and its</p>

				possible influence on results
Pick et al. [21]	<p>FND-seiz (n = 39), HCs (n = 42)</p> <p>Diagnosis/recruitment: as described in above section [12]</p> <p>Inclusion criteria: as described in above section [12]</p> <p>Exclusion criteria: as described in above section [12]</p> <p>Groups matched for: age, gender, ethnicity, handedness, and IQ, but FND-seiz group had higher HADS Anxiety and Depression, VOSP OD and WMS-III Family Pictures scores, relative to HCs</p>	<p>Emotional processing task: IAPS images (neutral, pleasant low arousal, pleasant high arousal, negative low arousal, negative high arousal), 6s exposure</p> <p>Primary dependent measures: subjective valence (negative-positive, 0-9) and arousal (low-high, 0-9) ratings, SCLs (baseline, task), SCRs (amplitude, frequency)</p> <p>Additional measures: HADS, seizure symptoms</p> <p>Cognitive tests: WASI, VOSP Object Decision, Family Pictures (WMS-III)</p>	<p>No significant group main effects on subjective ratings or frequency of SCRs</p> <p>In autonomic responders, SCR amplitude higher in FND-seiz group (n = 20) versus HCs (n = 23) (controlling for HADS Depression scores and medication)</p> <p>Positive correlation between SCR amplitude (negative/high arousal images) and (self-reported) ictal autonomic arousal symptoms (FND-seiz group)</p> <p>In autonomic non-responders, the FND-seiz group reported greater subjective negativity (for negative low and positive high arousal pictures) and increased subjective arousal (negative low arousal pictures), relative to HCs</p>	<p>Limitations as described in above section [12]</p>
Roberts et al. [22]	<p>FND-seiz group (n = 18) vs HCs high (n = 18) or low (n = 18) in post-traumatic symptoms (PTS)</p> <p>Diagnosis/recruitment: specialist epilepsy service, neurophysiologist/epileptologist</p> <p>Inclusion criteria: v-EEG evidence of FND-seiz, aged ≥ 18 years</p> <p>Exclusion criteria: psychosis, substance abuse, sensory impairment, epilepsy, uncertain</p>	<p>General affective images (IAPS stimuli), pleasant/unpleasant/neutral, 5s exposure</p> <p>Primary dependent measures: subjective ratings: intensity (high-low), valence (pleasant-unpleasant), emotional behaviours (e.g., facial expressions), ECG (cardiac inter-beat interval), HRV (respiratory sinus arrhythmia)</p> <p>Additional measures: PCL-S, SCL-90-R, DERS</p>	<p>No significant group effects for valence ratings</p> <p>FND-seiz group gave increased intensity ratings overall (vs both HC groups)</p> <p>Differences specifically for positive (vs PTS-low controls) and neutral (vs PTS-low and PTS-high HCs) images (controlled for medication effects)</p> <p>Fewer FND-seiz Ps showed positive behaviours to positive images (vs PTS-high HCs)</p>	<p>FND-seiz group scores on several SCL-90-R subscales higher than the PTS-low group, but influence on emotion processing task not explored</p> <p>Unclear whether comorbid neurological diagnoses (other than epilepsy)</p>

	diagnosis (FND-seiz), seizures / neurological disorder (HCs)		No group differences for ECG or HRV during affective image exposure, but FND-seiz group had reduced HRV at baseline (vs PTS-low HCs)	excluded from the FND-seiz sample
	Groups matched for: gender, age, years of education, income level, ethnicity, marital status, but FND-seiz group had higher scores on PCL-S, SCL-90-R Global Severity and subscales, plus DERS, compared to PTS-low HCs			
Seignourel et al. [23]	FND-movt (n = 12), HCs (n = 12)	IAPS stimuli (pleasant / unpleasant, neutral), exposure 6000ms, probe (white noise burst) 4200 / 5000 / 5800 post stimulus-onset	No significant group effects for subjective ratings of emotional responses	92% taking anti-depressants or anxiolytics – possible effects not explored in analyses
	Diagnosis/recruitment: neurologists, specialist movement disorder centre		FND-movt group showed potentiated startle responses for both positive and negative stimuli (HCs showed potentiation by negative stimuli and inhibition by positive stimuli)	25% FND-movt group (n = 3) had another comorbid movement disorder
	Inclusion criteria: Fahn & Williams' criteria	Primary dependent measures: subjective valence and arousal ratings, startle eyeblink response (latency, amplitude)	Startle amplitude did not correlate with BDI or STAI scores in either group	Potentially underpowered for subjective ratings
	Exclusion criteria: litigation, general medical, neurological or substance-related explanation for symptoms (FND-movt), psychiatric diagnosis, psychotropic medication, ongoing psychotherapy (HCs)	Additional measures: BDI, STAI		
	Groups matched for: age, education, gender, but FND-movt group had higher scores on BDI and STAI (state/trait)			
EMOTIONAL LEARNING & MEMORY TASKS				
Authors	Participants	Methods	Key findings	Limitations
Aybek et al. [24]	FND-par (n = 12), HCs (n = 13)	Personalised life event stimuli (escape, severe, neutral), visually presented statements (11s exposure)	No significant group effects on behavioural measures	Negative behavioural findings possibly related to small sample size

	<p>Diagnosis / recruitment: neuropsychiatrist, neurology/ neuropsychiatry outpatients</p> <p>Inclusion criteria: DSM-IV criteria, chronicity ≥ 2 years</p> <p>Exclusion criteria: psychosis, affective disorder, neurological disorder, non-fluency in English, MRI contraindication</p> <p>Groups matched for: age, gender, estimated IQ, autobiographical memory, HADS Anxiety scores, but HADS Depression scores higher in FND-par group</p> <p>Approximately 33% of each group medicated (e.g., antidepressants, analgesics)</p>	<p>fMRI, blocked design</p> <p>Primary dependent measures: reaction times (RTs for true / false responses), emotional distress caused by stimuli (Visual Analogue Scale, 0-10), BOLD response (whole-brain analysis, ROIs)</p> <p>Additional measures: Life Events and Difficulties Schedule (semi-structured interview), HADS, Autobiographical Memory Inventory</p> <p>Cognitive tests: NART – estimated IQ</p>	<p>Increased activation in FND-par group during escape-severe contrast in: right sensory motor cortex/SMA, right superior temporal cortex/insula/ temporo-parietal junction</p> <p>Decreased activation in FND-par group during severe-escape contrast in: left hippocampus/parahippocampal gyrus</p> <p>Significant after controlling for HADS Depression scores</p> <p>FND-par group showed increased left dlPFC activation during the escape condition (HCs)</p> <p>FND-par group showed reduced activity in inferior frontal cortex across conditions</p> <p>Greater connectivity between right SMA and amygdala/cerebellum in FND-par group across conditions</p>	<p>(statistically underpowered)</p> <p>No discussion of possible medication effects</p> <p>Lack of clinical comparison group</p>
Brown et al. [25]	<p>FND-par (n = 11), HCs (n = 28)</p> <p>Diagnosis/recruitment: neuropsychiatrist, neuropsychiatry inpatient & outpatient services</p> <p>Inclusion criteria: ICD-10 criteria for FND-par symptoms</p> <p>Exclusion: self-reported neurological, somatoform or major psychiatric disorder (HCs), non-fluency in English</p>	<p>Directed forgetting task: Word lists shown for 5s (neutral, negative), Ps directed to remember or forget</p> <p>Primary dependent measures: Immediate recall of 'remember' words, delayed unconditional recall after 10 min interference (all words to be recalled), intrusions (i.e. words not on lists)</p> <p>Cognitive tests: Autobiographical Memory Interview, Logical Memory (WMS-III), Trail-Making</p>	<p>Immediate recall: FND-par group recalled fewer words than HCs, particularly for 'remember' words</p> <p>Delayed recall: FND-par group recalled fewer words, particularly for 'remember' words, but no valence (negative/neutral) by group interaction</p> <p>HADS Depression scores did not correlate with primary dependent variables, including memory for negative words</p>	<p>Controls not screened for medication/ psychiatric disorder</p> <p>Most FND-par patients taking psychotropic medication</p> <p>Inclusion of 11 FND-par patients in memory task possibly underpowered</p>

	Groups matched for: age and gender, but HCs had significantly lower HADS Anxiety and Depression scores, and higher NART scores, relative to FND-par group	Task (set-shifting, processing speed), Stroop Colour-Word, NART Additional measures: HADS		Medication effects not excluded
Kranick et al. [26]	FND-movt (n = 20); HCs (n = 20) Diagnosis/recruitment: specialist neurological service Inclusion criteria: DSM-IV-TR criteria, clinically definite FND-movt, ≥ 18 years Exclusion criteria: significant medical or neurological disorder Groups matched for: age and gender, but FND-movt group had higher scores on BDI and STAI	Conditioned facial expressions (happy, fearful, neutral) to tones Tones used in a 'binding' experiment - participants judged the timing of key presses (action) with the occurrence of the tones (effect) Primary dependent measures: verbal reports of perceived clock position (key press or tone presentation) Additional measures: BDI, STAI	Reduced tone binding in the FND-movt group relative to HCs (controlling for elevated BDI / STAI scores) No significant effects of valence of faces	Possible medication effects not explored Non-clinical control group only No examination of acquisition of conditioning with faces
Morris et al. [27]	FND (mixed symptoms) (n = 25); HCs (n = 20) Diagnosis/recruitment: neurologist/neuropsychiatrist, specialist FND service Inclusion criteria: FND symptoms included pain, motor, sensory and seizures Exclusion criteria: age < 18 years, neurological disorder, MDD (severe), bipolar disorder, psychosis, substance abuse Groups matched for: age, gender	Associative conditioning of neutral stimuli (abstract shapes) with aversive or neutral IAPS images and sounds Aversive operant conditioning (including previously conditioned shapes), with choices leading to variable probabilities of monetary loss fMRI Primary dependent measures: response accuracy, trials to acquisition, ROI (amygdala, dIPFC), functional connectivity	FND group less accurate in operant conditioning responses and showed more trials to acquisition FND group showed impaired learning for the aversively conditioned stimulus only Increased activity in left amygdala and trend towards reduced engagement of dIPFC during loss in FND group relative to HCs Increased resting functional connectivity of bilateral amygdala and right dIPFC in FND, relative to HCs	Majority of FND sample medicated, but possible medication effects not explored Non-clinical control group Tertiary recruitment setting

Additional measures: STAI, BDI-II, MINI

Key: AB = attentional bias; AEDs = antiepileptic drugs; AT = Animations Task; BAI = Beck Anxiety Inventory; BDI = Beck Depression Inventory; BFRT = Benton Facial Recognition Test; BOLD = blood oxygenation level dependent; CNS = central nervous system; DASS = Depression Anxiety and Stress Scale; DERS = Difficulties in Emotion Regulation Scale; dlPFC = dorsolateral prefrontal cortex; DSM-5 = Diagnostics and Statistical Manual of Mental Disorders – 5th edition; ECG = electrocardiography; ECs = epilepsy controls; EEG = electroencephalography; ERQ = Emotion Regulation Questionnaire; ET = essential tremor; ETI = Early Trauma Inventory; fMRI = functional magnetic resonance imaging; FND-seiz = functional neurological disorder – seizure symptoms; FND-movt = functional neurological disorder – abnormal movements; FND-par = functional neurological disorder – paralysis/paresis; FSS = functional somatic symptoms; GFP = global field power; HADS = Hospital Anxiety & Depression Scale; HAM-A = Hamilton Anxiety Rating Scale; HAM-D = Hamilton Depression Rating Scale; HCs = healthy controls; HRV = heart rate variability; IAPS = International Affective Picture System; IASC = Inventory of Altered Self Capacities; ICD-10 = International Classification of Diseases – 10th edition; IFG = inferior frontal gyrus; IQ = intelligence quotient; LEAS = Levels of Emotional Awareness Scale; LoC = loss of consciousness; MADR = Montgomery-Asberg Depression Rating Scale; MASC = Movie for the Assessment of Social Cognition; MCs = medical controls; MDD = major depressive disorder; MEG = magnetoencephalography; min = minute; MINI = Mini International Neuropsychiatric Interview; MRI = magnetic resonance imaging; ms = milliseconds; MSS = Mental States Stories; NART = National Adult Reading Test; OD = organic dystonia; Ps = participants/patients; PAG = periaqueductal grey; PANAS = Positive and Negative Affect Schedule; PCL-S = PTSD Checklist – specific; PFC = prefrontal cortex; POMS = Profile of Mood States; PSS = Perceived Stress Scale; PSSI = Post-traumatic Stress Scale Interview; PTS = post-traumatic symptoms; PTSD = post-traumatic stress disorder; RME = Reading the Mind in the Eyes; ROI = region of interest; RT = reaction time; s = seconds; SCID = Structured Clinical Interview for DSM; SCR = skin conductance response; SCL = skin conductance levels; SCL-90 = Symptom Checklist – 90; SDQ-20 = Somatoform Dissociation Questionnaire – 20 item; SES = socioeconomic status; SF-36 = Short-Form Health Survey – 36 item; SMA = supplementary motor area; STAI = State Trait Anxiety Inventory; TAS = Toronto Alexithymia Scale; TBI = traumatic brain injury; TEC = Traumatic Experiences Scale; TENS = transcutaneous electrical stimulation; ToM = Theory of Mind; v-EEG = video-electroencephalography; vmPFC = ventromedial PFC; VOSP OD = Visual Object and Space Perception – Object Decision; WASI = Wechsler Abbreviated Scale of Intelligence; WM = working memory; WMS-III = Wechsler Memory Scale – third edition; YoE = years of education

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